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## **Headgear-induced temporary pressure alopecia**

**Catherine Liu, Faisal Abbas, Jadbinder Seehra**

### **Introduction**

Alopecia encompasses a range of types of hair loss and can be induced due to the presence of aberrant pathology, trauma and contact of the scalp with chemical toxins (Abel and Lewis, 1960). Specifically, pressure alopecia is classified as non-scarring hair loss localised in a defined single area or multiple areas of the scalp, usually attributed to a mechanical cause (Leonardi et al., 2008; Zuehlke et al., 1981). Both an immunologic and genetic aetiology have been suggested (Zuehlke et al., 1981). However, pre-disposing factors also implicated include periods of intra- and post-operative immobility (Abel and Lewis, 1960; Domínguez-Auñón et al., 2004; Wiles and Hanson, 1985; Lawson et al., 1976) and iatrogenic causes such as the use of intra-operative devices (Iwai et al., 2009; Matsushita et al., 2011) resulting in excessive or chronic trauma to the hair of the scalp (Abel and Lewis, 1960).

Pressure alopecia can be distinguished from most other types of hair loss by clinical history and presentation. The localised, patchy appearance of pressure alopecia differentiates it from androgenetic alopecia, telogen effluvium, and anagen effluvium, and its non-cicatricial nature contrasts to hair loss occurring after trauma, lupus or lichen planus. Histological investigation cannot always discriminate between other forms of localised nonscarring alopecia (alopecia areata, chronic traction areata and trichotillomania), but the patient history is often diagnostic (Premkumar and Vidya, 2013; Eudy and Solomon, 2006).

Within the United Kingdom, there is an increasing demand for orthodontic treatment in young children. In 2013, Rolland et al. (2013) reported that 9% and 18% of 12- and 15-year-olds respectively were undergoing some form of orthodontic treatment. As part of fixed appliance therapy, extra-oral devices such as headgear are sometimes used to manage anchorage demands or facilitate distal movement of the upper posterior teeth. Headgear consists of an extra-oral unit which provides anchorage, a force delivery system, an intermediate component (facebow) and intra-

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oral attachments. The force delivery (snap-away) module also incorporates a safety feature to reduce the risk of recoil injuries; however, both soft tissue and ocular injuries have been reported with the use of headgear (Samuels and Jones, 1994).

The development of hair loss associated with the use of headgear is a rare phenomenon (Leonardi et al., 2008; Zuehlke et al., 1981; Eudy and Solomon, 2006). The development of alopecia in adolescence can result in a negative impact on both emotional and psychological well-being (Christensen et al., 2017).

This clinical case report describes the development of bilateral temporary pressure alopecia in a young patient undergoing orthodontic treatment involving the use of headgear.

## **Case report**

A 13-year-old Ecuadorian male was referred by his general dental practitioner to the Orthodontic Department at Kings College Hospital to address his complaint of dental crowding. He was medically fit and well with no history of allergies.

The patient had a Class I skeletal pattern with increased vertical proportions complicated by mandibular asymmetry with the chin point to the right-hand side, crowding of the arches and an anterior displacement on mandibular closure. In occlusion, the incisor relationship was Class III with reduced overjet and overbite, and a lower centreline shift of 2mm to the right. The molar relationships were a full unit Class II on the right side and Class I on the left side. Both the UR2 and UL2 were in crossbite and moderate-to-severe crowding was present in both the upper and lower arches (Figure 1 and Figure 2).

During the initial orthodontic consultation, photographic records and impressions for study models were taken, as well as a pretreatment lateral cephalometric radiograph (Figure 3) and orthopantomogram (Figure 4). An impacted unerupted upper right second premolar was identified on the radiographs. The cephalometric values at baseline were as follows: SNA 81°, SNB 79°, ANB 2°, Upper incisor –Maxillary plane 113°, Lower incisor to Mandibular plane 94°, Maxillary Mandibular Plane Angle 33° and Lower Anterior Face Height ratio 57%.

The proposed treatment plan aimed to address the patient's concerns, relieve the crowding, correct the centrelines and achieve a Class I occlusion. This involved the use of high-pull headgear to distalise the upper first permanent molars and provide vertical anchorage control, the loss of four premolar units and upper and lower fixed appliances (Pre-adjusted edgewise appliance, 0.022"x0.028" bracket slot with MBT prescription). The headgear was fitted with a safety release mechanism, exerting a force level of 350 grams bilaterally, and to be worn for 10-12 hours every day.

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At the patient's first review appointment 4 weeks later, the patient presented complaining of tenderness and hair loss in the right and left temporal regions. Clinically, the areas of hair thinning were directly in relation to where the safety release modules of the headgear contacted the patient's scalp (Figure 5). A differential diagnosis of contact allergy or thermal trauma to the hair follicles was proposed. The force level was remeasured and the patient was reassured.

At the subsequent appointment 8 weeks later, the patient complained of further hair loss bilaterally in the temporal regions (Figure 6), and reported he was only wearing the headgear every other day due to his concerns over hair loss. Furthermore, he had been experiencing teasing and bullying at school as a result of these areas of hair loss. On examination, the areas of the scalp in contact with the safety release modules showed irregularly shaped patches of hair loss. The patient was advised to stop wearing the headgear and a transpalatal arch was fitted to reinforce the anchorage.

On review 4 weeks later, the areas of hair loss had begun to resolve (Figure 7). After a further 4 weeks, hair density in the temporal regions had returned to normal (Figure 8); a diagnosis of temporary pressure alopecia was confirmed. The patient did not experience any further symptoms or hair loss. Written informed consent was obtained from the patient and parent for publication of patient information and images.

## **Discussion**

The occurrence of chronic hair loss due to pressure-induced tissue hypoxia is rare and there have been only four reported cases of pressure alopecia associated with orthodontic treatment in the existing literature.

Leonardi et al. (2008) described a 14-year-old male who experienced bilateral alopecia in the parieto-occipital areas of the scalp which began 4 months following commencement of combi-pull headgear. A force level of 500 grams per side was worn for 12-14 hours a day. Following discontinuation of headgear wear regrowth of the hair occurred. Similarly, Zuehlke et al. (1981) reported a case of a 7-year-old female suffering localised hair loss 2-3 weeks after wearing straight-pull headgear for 14-18 hours a day at an unspecified force. Rather than ceasing treatment, a sponge pad was incorporated between the headgear strap and the scalp, and within 5 months hair density had returned to normal. Premkumar and Vidya (2013) described a 12-year-old male with cranial asymmetry who, after wearing high-pull headgear at 450 grams per side for 12-14 hours a day for 5 months, developed pressure alopecia on the left side of the scalp. Successful management was achieved with benzoic acid, multivitamins and altered force application (reduction of force to 300 grams on the left side) and hair regrowth occurred after 6 weeks.

Zuehlke et al. (1981) also described a 13-year-old female who was wearing high-pull headgear at 340 grams per side for 12-14 hours a day without problems, but developed patches of alopecia and soreness at the four pressure sites when this was increased to 700 grams per side for 20 hours a day. Unlike the previous cases, even after discontinuing treatment with headgear two more patches appeared within the next month. Interestingly this patient's sister had a history of alopecia areata. Management involved intralesional triamcinolone acetonide, and 6 weeks later hair regrowth began, and within 5 months hair density was fully recovered. In comparison

to these cases, the headgear force used in this case was much lower in magnitude. The patient also had no family history of alopecia. However, he still developed this condition which reassuringly resolved following removal of the source of trauma to the hair follicles.

The majority of case reports of pressure alopecia have been observed following extended surgical procedures, giving rise to the alternative name of postoperative alopecia (Abel and Lewis, 1960; Domínguez-Auñón et al., 2004; Wiles and Hanson, 1985; Lawson et al., 1976; Iwai et al., 2009; Matsushita et al., 2011; Huang et al., 2018; Chang et al., 2016; Bagaria and Luck, 2015). Other causes of pressure alopecia reported in the literature include electroencephalogram electrodes (Kunapareddy et al., 2018; Morris et al., 1992), headbands (Sano et al., 2018), fixed head positioning whilst watching television (Thiem et al., 2016) and breakdancing (Bonifazi, 2006). The first reported cases of postoperative pressure-induced alopecia were documented by Abel et al. in 1960; eight patients underwent gynaecological procedures lasting between 4 and 8 hours. Between 3 and 28 days later, they all then experienced alopecia over the vertex, where the head was rested during the operation. Five out of the eight also suffered swelling and exudate from the vertex which resolved in 1 week, and all the patients' hair began to regrow in 4 to 12 weeks. There was no significant correlation between occurrence of symptoms and duration of operation. Since this case series was published, there have been numerous reports of similar findings of temporary pressure alopecia after various surgical procedures including orthognathic operations (Iwai et al., 2009; Matsushita et al., 2011). There have been fewer reports of permanent pressure alopecia, which was defined as alopecia that did not resolve within 1 year; Lawson et al. (1976) found a close correlation between the duration of intubation and the likelihood of permanence.

Postoperative pressure-induced alopecia has been most well reported and attributed to immobilisation during long operations with prolonged pressure exerted on a circumscribed area of the scalp. Onset is most frequently 1 month after surgery, with hair regrowth within 3-5 months. However, if the pressure is severe or prolonged then the consequent alopecia may become permanent and even cicatricial, with highest risk when pressure duration exceeds 24 hours. In these severe cases the patient may also experience pain, oedema and central ulceration in the affected area (Leonardi et al., 2008; Kunapareddy et al., 2018; Morris et al., 1992; Davies and

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Yesudian, 2012; Bruce et al., 2002; Goodenough et al., 2014; Boyer and Vidmar, 1994).

The mechanism of pressure alopecia has been explained by the occlusion of blood vessels in the scalp causing localised ischaemia, which impedes normal follicle activity. This results in loosening of the follicle and later epilation of the hair (Davies and Yesudian, 2012; Boyer and Vidmar, 1994). When normal circulation is restored, follicle activity and thus hair growth resumes (Abel and Lewis, 1960). More severe or prolonged pressure leads to greater hypoxia, followed by tissue necrosis and permanent follicular loss.

Duration of contact appears to be the main factor associated with risk of pressure alopecia (Wiles and Hansen, 1985), but other mechanisms are likely to also be interacting since pressure alopecia can develop even after shorter durations of pressure (Loh et al., 2015; Lee et al., 2012) and even outside contact areas (Morris et al., 1992). Zuehlke et al. (1981) postulated that a genetic predisposition was an essential factor, and that pressure was merely a trigger. A recognised exacerbating factor is hypotension as this causes hypoperfusion of the scalp; this may be due to hypotensive anaesthesia, peri-operative blood loss, or surgical positioning such as in the Trendelenburg position used in some gynaecological procedures (Abel and Lewis, 1960; Lee et al., 2012; Patel and Henschel, 1980). Other risk factors include psychosomatic comorbidities (Misery and Rousset, 2001; Picardi et al., 2003; Ruiz-Doblado et al., 2003), female gender, and cardiac abnormalities (Davies et al., 2012; Goodenough et al., 2014).

Pressure alopecia is most commonly confused with alopecia areata, a much more prevalent condition (Caldwell et al., 2017) with an unclear genetic and immunological aetiology (Zuehlke et al., 1981). It presents as one or more rounded patches and exhibits clinical phases of activity, inactivity and regrowth (Eudy and Solomon, 2006). Unlike pressure alopecia, alopecia areata has no prodromata; pressure alopecia however follows prolonged pressure to an area of the scalp and may be preceded by erythema, swelling, exudate, crusting and tenderness (Abel and Lewis, 1960; Wiles and Hansen, 1985; Chang et al., 2016; Bagaria and Luck, 2015). Abel and Lewis (1960) also reported faster and prompter hair regrowth in areas of pressure alopecia compared to alopecia areata.



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In contrast to other types of alopecia, pressure alopecia does not appear to respond to pharmacological treatments. Examples from the literature suggest that cortisone and vitamin B (Leonardi et al., 2008), erythromycin (Wiles and Hansen, 1985), and carpronium chloride (Iwai et al., 2009), do not have any beneficial effect on hair regrowth. The most effective management of pressure alopecia entails identifying the cause as early as possible and removing or reducing the causative agent (Zuehlke et al., 1981). Buffering the pressure on the scalp for example using a sponge pad, frequently moving the head position during prolonged surgery, or scalp massages have been suggested to prevent the development of pressure alopecia (Matsushita et al., 2011). Lawson et al. (1976) found that peri-operative rotation of the head every 30 minutes reduced risk of pressure alopecia from 14% to 1%. A prospective study performed by Huang et al. (2018) showed that using an alternating inflating head pad during open heart surgery significantly lowered incidence of postoperative alopecia compared to a conventional gel head pad.

An alternative conventional method to reinforce anchorage e.g. Transpalatal arch was used following the complications incurred with extra-oral headgear. Based on moderate quality evidence level, skeletal anchorage devices appear to be more effective than conventional methods (Jambi et al. 2014). However, after considering the clinical situation, the social circumstances and the further anchorage demands, a transpalatal arch was deemed appropriate. As highlighted in this clinical case, it is important to consider the psychosocial effect on the patient. The patient experienced teasing from his peers as a result of the localised hair loss, eliciting a negative emotional and psychological effect. With a large proportion of children and teenagers undergoing orthodontic treatment (Rolland et al., 2013) it should be remembered that this age group can be particularly susceptible to peer victimisation (Seehra et al., 2011). Alopecia areata has been found to increase the sufferer's exposure to bullying, significantly impair their health-related quality of life, and negatively impact their emotional and psychological wellbeing (Christensen et al., 2017; Rencz et al., 2016). Therefore, since the clinical presentation is almost identical, it can be assumed that pressure alopecia potentially affects the sufferers in a similar detrimental way. For this reason, a patients and their parents could be expected to attach significance to the risk of pressure alopecia, rendering it a material risk of orthodontic treatment (Montgomery v Lanarkshire Health Board, 2015). Clinicians

would therefore be justified in presenting this risk to patients if using headgear as part of the treatment (Leonardi *et al.*, 2008) when obtaining informed consent.

## **Conclusions**

In addition to the standard risks associated with orthodontic treatment (root resorption, damage to the tooth surface, periodontal damage, orthodontic instability, hypersensitivity to certain materials, and soft tissue injuries) as summarised by Travess *et al.* (2004), headgear conveys additional complications, including serious eye injury (Samuels and Jones, 1994), and difficulty performing everyday activities such as eating and sleeping (Premkumar and Vidya, 2013). Although the incidence of headgear-induced pressure alopecia is low, the potential impact on the psychological wellbeing of patients is significant and therefore should be considered as a risk to discuss and consent for prior to commencing headgear treatment (Leonardi *et al.*, 2008).

In this rare case of pressure alopecia resulting from orthodontic headgear, it was fortunate that no permanent damage to the hair follicles occurred. Treatment using headgear was discontinued as the patient's psychosocial well-being was being negatively affected. The patient was reviewed regularly to assess hair regrowth and confirm the diagnosis of temporary pressure induced alopecia. The patient was not referred to a dermatologist because the condition resolved completely following cessation of headgear wear; however, had the alopecia persisted then a dermatology opinion would be prudent.

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